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MUSCULAR DYSTROPHY OF BEEF CALVES IN THE NORTH OF SCOTLAND

BY

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INTRODUCTION

In the spring of 1951 it was evident that farmers in Inverness and adjoining counties were experiencing more trouble than usual when they turned their beef calves out from their winter quarters to grass for the first time. Sudden deaths and stiffness were their main cause of worry. The unusually protracted and severe winter of that year was thought to be responsible for the increased prevalence of these conditions.

In the autumn, at a meeting of the North of Scotland Division of the British Veterinary Association in Forres, Mr. Suttie and I initiated a discussion on the cases seen in the spring, for the purpose of finding if there was a common factor. It became obvious that similar symptoms had been seen each spring for many years throughout the northern beef rearing area from Caithness to Kincardine.

A description of the symptoms was given in the annual report of the V.I.O. for the North of Scotland which is circulated amongst the other V.I.Os. in Britain. The description was thought by A. L. Wilson of the West of Scotland College of Agriculture to be highly suggestive of Muscular Dystrophy, which he had seen at the Hannah Dairy Research Institute where it had been reproduced experimentally by Dr. K. L. Blaxter, and which had been shown to be present in field cases of stiffness in the south of Scotland by Dr. J. T. Stamp and Dr. Blaxter in 1951. In the spring of 1952, with the help of Dr. Blaxter, and thanks to the co-operation of local veterinary surgeons who made all clinical cases available for examination, it was possible to show that both the stiff cases and the sudden deaths were manifestations of muscular dystrophy.

HUSBANDRY

The Moray Firth coastal area and the counties of Aberdeenshire and Kincardineshire consist of mixed farms, large and small. Aberdeen Angus calves pure or crossed with Shorthorn, are bred on most of the farms irrespective of their size.

The in-calf cows are taken indoors in October or November and are housed until late April or May. During this time they are kept in good condition mainly on straw and turnips. Up to 10 lb. per day of hay and up to 2 lb. per day of oats are sometimes fed from calving onwards. The cows usually calve between December and May.

The calves suckle their dams, normally one calf to a cow, but on some farms an extra calf is bought for each cow. The calves may be tied individually or confined together in pens. In all these systems they are released twice or thrice daily to suckle the cows, which are tied in double standings. On a few farms the cows calve in courts so that calves have free access to their dams at all times. Many calves receive their dams' milk only, but some may, in addition, be offered hay from six weeks of age.

It has long been recognised in this area that calves are extremely susceptible to stiffness and lameness when put out to grass for the first time. Then, too, sudden deaths are regrettably common. In attempts to minimise these risks, it has become accepted practice to introduce calves gradually to increased exercise, and to the relatively cold temperature of outdoors, by admitting them to a cattle court vacated by winter fattening bullocks or store bullocks, for a week or more before turning the calves out. During this period the cows are out at grass for part of the day. These precautions, however, have not prevented the stiffness or the sudden deaths.

CLINICAL DESCRIPTION

Several forms of the disease have been seen in the field, all of which can be attributed to muscle abnormalities. The signs observed are identical with those observed at the Hannah Dairy Research Institute in calves suffering from dietary deficiency of vitamin E (Blaxter, Watts & Wood, 1951[1] and 1952[2]). This fact alone suggests that vitamin E is involved in the aetiology of the disease. By far the most common syndrome is stiffness of the shoulder region. Stiffness affecting the muscles of one or a combination of the following sites has also been seen : dorsal muscles of the neck, the muscles of the back, thighs, and quadriceps group, and the intercostal muscles. In addition signs of diaphragmatic insufficiency (confirmed by finding lesions of muscular dystrophy at autopsy) have been observed. The stiffness has always been found to be bilaterally symmetrical.

Cardiac signs have been recognised and these usually precede sudden death, which has been shown to be associated with degeneration of the myocardium. Some affected calves show these signs, while others show no noticeable signs of illness whatsoever and yet suddenly succumb.

In a very few cases difficulty in swallowing has been noted, and in these the muscles involved in swallowing have shown lesions of muscular dystrophy. More detailed descriptions of these various types of the disease are given below.

Stiff Type

Usually the fore limbs are the first to show stiffness. The calf rises with difficulty and walks reluctantly. Once under way, it carries its fore limbs forwards and outwards and takes slow steps. The line of the forearm comes back towards the vertical but not beyond. In this type the chest is usually dropped, so that the line of the back descends from the root of the tail between the scapular cartilages which protrude above the line of the back. The head is carried low. At rest such calves will stand with their limbs spread out. If the back muscles are gently grasped with one hand the animal will hollow its back.

In the typical "shoulder" case the muscles which normally support the chest, namely the trapezius and rhomboideus muscles and the superficial pectoral muscles are affected. The thorax, thus losing its normal support, drops, and the scapular cartilages protrude above the line of the back. Often they press hard against one another when the animal is standing.

Frequently one or more calves on a farm are found one morning to be so stiff that they are incapable of rising. Such animals retain their appetites and soon learn to suckle their dams from a sitting position. The casual observer might assume that such calves are perfectly normal, until attempts are made to make them rise.

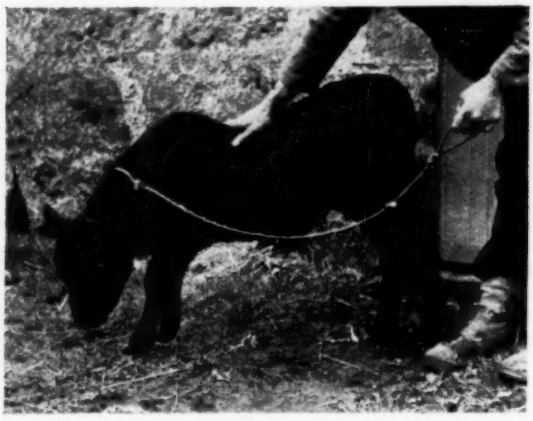

Fig. 1.—Hollow back.

Fig. 2.—Limbs spread.

Fig. 3.—Limbs spread.

The affected muscles usually retain their normal bulk. Occasionally, however, there may be hypertrophy. In such a case it is possible to determine loss of elasticity in the muscles by palpation. In other cases, however, it is very difficult to decide by palpation whether or not a muscle is dystrophic.

Fig. 4.—Hypertrophy of shoulder muscles (enlarged carpal joint is incidental).

A great variety of gait abnormalities have been observed. This is only to be expected when it is realised that almost any combination of limb muscles may become dystrophic. Cases have been seen where the hind limbs alone have been affected, resulting in a dragging of the toes, or where the longissimus dorsi have shown marked hypertrophy so that the back has become arched.

Respiratory Type

Rigidity of the chest is a feature of dystrophy of the intercostal muscles. This gives rise to abdominal breathing. Where the diaphragm is affected, exaggerated movement of the chest wall is seen, suggestive of pneumonia. It is seldom, however, that either chest or diaphragm types are uncomplicated. A combination of both, further complicated by dystrophy of heart muscle or abdominal wall, gives a very confused clinical picture. Fast, distressed breathing, with a rapid, sometimes irregular, pulse, and normal temperature, constitute as accurate a description as can be given of such cases. Again, the affected calf usually retains its appetite unless distress is severe.

Cardiac Type

The animal is reluctant to stand or is unable to do so. The pulse is extremely fast and weak (180 per minute). Enlargement of the heart may sometimes be determined clinically. If the floor of the abdomen, caudal to the costal arch, is raised with both hands a grunt may be provoked. Discomfort is due presumably to pressure on the congested liver or to indirect pressure on the heart. The temperature is normal or sub-normal though "sweating" is occasionally seen. In advanced cases weakness is extreme. The blood pressure may be so low that the jugular vein cannot be raised. Undue handling may precipitate death. Recovery is not uncommon in cases which merely show accelerated respiratory and pulse rates, combined with weakness of the limbs. Advanced cases die.

After exertion, which could be that of suckling, the heart case may stand with its head down, breathing very rapidly, fall down in a coma and die, death following one to two minutes after the onset of distress. Very often the calf has appeared to be perfectly normal until then.

If the muscles of swallowing are affected the suckling time increases. Appetite is retained but the ability to feed becomes less. Inhalation pneumonia almost invariably sets in. Difficulty in raising the head to suckle is also experienced where the dorsal muscles of the neck are affected.

Incidence

This varies strangely within the area. A district may have many affected farms one year and none the next, whereas in the same years the reverse may be true of a neighbouring district. Then, with no apparent change in management, a farm may experience a gradually increasing familiarity with the disease. On one farm in the Nairn area, with 40 spring calves, the farmer reports that there was no history of the disease until 1951 when six cases were experienced. In 1952 nearly all the 40 calves born were affected and 50 per cent. died. On many farms only one calf is affected, but so far it has been difficult to forecast when a disaster of the magnitude just described will occur. Surveys and experience suggest that roughly 25 per cent. of the calf-rearing farms in the area experience the disease.

The disease has been known for at least 50 years in the north of Scotland. The calves are known locally among farmers as " crochly " calves and, in the past, the condition has been diagnosed as rheumatism, abomasal obstruction or " hairball " (see N.V.M.A. handbook on calf diseases, 1947, for similarity of " hairball " to muscular dystrophy), rickets, indigestion and pneumonia.

It is most unusual for a calf born before mid-January to be affected. February, March and April born calves are the most susceptible.

Most cases occur in the first few days at grass, but fresh cases do occur up to four weeks after the calves are turned out. Occasional cases of stiffness occur through the summer at grass. The stiff type of case has also been encountered while still indoors. When such cases are found it is likely that further cases will occur when the calves go out to grass.

Heart cases are quite common before cows or calves go outside. The exertion of suckling precipitates an attack. It has been suggested that more heart attacks occur on Sundays than on other days of the week. This is presumably because calves are suckled early on Saturday evenings and late on Sunday mornings with consequent excitement and exertion on the part of the calves.

Post-Mortem Examination

Macroscopical

Severely affected muscles are obviously pale and non-translucent, having the creamy white appearance of cooked chicken breast. They do not have the elasticity of normal muscles when squeezed gently between the fingers, but feel " wooden. " In many cases when a piece of muscle is placed on paper and left for an hour or so, straw-coloured fluid will be found surrounding it. The muscle lesions are bilaterally symmetrical.

In typical " shoulder " cases the superficial pectoral, rhomboideus and trapezius muscles are affected. The supraspinatus and the infraspinatus may also be involved, but almost any skeletal muscle may be affected. Lesions have been commonly observed in the muscles of swallowing, the longus colli, the longissimus dorsi, the quadriceps group and the posterior muscles of the thigh. Lesions have also been observed in intercostal muscles, diaphragm and left and right ventricles. Muscles of the gaskin and the forearm are seldom affected.

Examination of a calf which has died suddenly from affection of the heart constantly shows passive congestion of the liver which is enlarged, purple and has rounded edges. Most of the body lymph nodes are congested. Yellow bands are usually plainly visible in the hypertrophied ventricle walls, and the loss of elasticity can be appreciated with the fingers. Occasionally the ventricle walls appear normal to the naked eye but dystrophic lesions may be demonstrated histologically. Again the field disease appears closely comparable to that produced experimentally.

Histological

All the stages described in experimental muscular dystrophy and cod liver oil poisoning have been observed (MacDonald, Blaxter, Watts & Wood, 1952[3]). In the experimental disease the sequence of events is as follows—swelling of individual fibres, increased affinity for eosin (at this stage transverse striations are still present) hyaline degeneration, and the replacement of fibres by nuclei thought to originate from the sarcolemma. A severely affected muscle is seen to consist almost entirely of these nuclear masses. It is probable that this sequence occurs in field cases also.

Biochemical

Affected skeletal muscles from cases in the north of Scotland have been shown at the Hannah Dairy Research Institute to have abnormally low creatine contents. Figures of the order of 80 mg. Creatine/100 grammes wet muscle were obtained compared with figures of 350–500 mg. per 100 grammes for normal muscles. Both figures might well have been obtained from the same calf, one from a dystrophic muscle and one from a normal muscle.

The tocopherol content of the livers of dead calves was low.

Experimental Prevention

It was shown in the spring of 1953 that the disease could be prevented by feeding vitamin E in the form of alpha tocopheryl acetate to cows before calving, and to their calves from birth until they went to grass. Feeding of vitamin E to the calves alone also prevented the disease. (Blaxter & Sharman, 1953[4], 1954[5].)

Treatment

Roughly 60 per cent. of the " stiff " cases which occur when the cows are turned out will recover without treatment provided they are kept warm and are not tempted to indulge in exercise. Recovery is, however, slow. On one farm near Thurso in 1951, nine calves out of a total of eleven were found one morning to be unable to rise. They were taken inside in a wheel-barrow and their dams were manoeuvred over the calves for suckling so long as the calves were unable to rise. Of the nine calves two died and seven recovered. Complete recovery took about a month. No other treatment was adopted.

Using alpha tocopheryl acetate in tablet form by mouth, early cases can confidently be expected to show signs of recovering in a day or two. The dose now used in this area is 600 mg. as an initial dose followed by 200 mg. once daily until recovery is complete. Exercise is, of course, restricted and calves are kept in on cold or wet days, and every night, until recovery is complete. In some cases the response to treatment is spectacular.

The drug was given by intramuscular injection at the Hannah Dairy Research Institute but was found not to be readily absorbed from the injection site. Thereafter the oral route of administration was adopted.

Discussion

Having recognised the field disease as muscular dystrophy it was assumed to be associated with deficiency of vitamin E in the rations of the dams. As it is known that considerable placental transfer of vitamin E takes place (Parish, Wise, Latschar & Hughes, 1950[6]) it was considered that calves born before January would have received considerable stores *in utero* from autumn grass while later-born calves would have depleted stores. Wide variations in vitamin E stores had been noted in the west of Scotland and had been shown to be due to variations in the dams' rations (Blaxter, Brown & MacDonald, 1953a[7]). Colostral and milk transfer of vitamin

E would be negligible to late-born calves where the dams' rations during the later part of pregnancy consisted of straw and turnips which have low vitamin E contents. Adequate supplies of vitamin E are essential for construction and maintenance of normal muscle ; without vitamin E, lesions of muscular dystrophy develop. (Blaxter, Watts & Wood, 1952[2].)

TABLE I

THE FOLLOWING ANALYSES OF FOODSTUFFS FROM THE INVERNESS AREA WERE CARRIED OUT AT THE HANNAH DAIRY RESEARCH INSTITUTE

	Total Tocopherol Contents (average figures) mg. per 100 grammes DM.
Hay	2·5
*Artificially Dried Grass ...	2·5 one sample only
Barley Straw	4·7
Oat Straw	2·7
Bruised Oats	1·5
Yellow Turnips	1·5
Swede Turnips	1·3
Grass Silage	30·0
Wet Distiller's Grain ...	3·5

* A poor-quality sample. For dried Cocksfoot, Brown quotes figures of 17–24 mg. per 100 grammes DM (Brown 1953[3]).

TABLE II

COW WINTER RATIONS TYPICAL OF THE NORTH OF SCOTLAND WITH THEIR TOTAL TOCOPHEROL CONTENTS

	Food	Daily Consumption kg.	Daily Consumption lb.	Per cent. Dry Matter	Daily Tocopherol intake mg.
Ration (1)	Oat Straw	9·1	20	86·0	362
	Swede Turnips	36·4	80	9·3	44
					406
Ration (2)	Hay	4·6	10	86·0	98
	Barley Straw	4·6	10	86·0	204
	Bruised Oats	0·9	2	87·0	12
	Yellow Turnips	27·3	60	9·3	39
					353

It may be computed that at pasture in summer a cow may eat 120 lb. of grass in a day, which contains at least 3 grammes total tocopherol. The greatly reduced winter intake is obvious from the above table. As far as vitamin E activity is concerned the reduction may be even greater since many of the feeds analysed contained low proportions of the biologically most active tocopherol, alpha tocopherol (Blaxter & Brown, 1952–53[9]).

As it is well known in the area that many cases appear after a cold, windy night, it is assumed that the clinical disease is precipitated by increased exercise and by cold. When it is considered that affected muscles consist of inelastic material composed of sarcolemmal nuclei it is not surprising that severe lameness follows exercise.

Diagnosis of the disease is made from the history, the clinical findings and *post-mortem* examination. Liver tocopherol and muscle creatine values are useful aids to diagnosis. In addition, if cows and calves are still indoors the vitamin E status of the herd may be assessed on blood serum vitamin E contents. From the results of our joint experiments figures of below 70 μg. total tocopherol per 100 ml. in calves and below 150 μg. total tocopherol per 100 ml. in cows indicate a low vitamin E status. (Blaxter & Sharman, 1954[5].) It must be emphasised that there is wide variation in serum tocopherol levels among individuals in commercial herds, so that single samples are worthless. Again, serum values are only of use where cattle are not ingesting foods which are rich in vitamin E (*e.g.* grass), as tocopherols quickly gain access to the blood-stream.

While we have shown that dosage with vitamin E capsules or tablets will prevent the disease, clearly an endeavour should be made to devise rations for in-calf cows which will supply adequate amounts of vitamin E in the diet. Grass is our most readily available source of vitamin E. Silage-making only reduces the vitamin E content of grass by 9 per cent. but hay-making reduces the vitamin E content by 95 per cent (Brown, 1953[3]). On the farm mentioned earlier as having had 40 cows with their 40 calves and 50 per cent. calf mortality in 1952, extra grass was sown in the spring of 1952 and grass silage was made from this young grass. It was of good quality and contained the equivalent of 30 mg. alpha tocopherol per 100 grammes DM. It was fed at the rate of 20 lb. per day to the cows throughout the winter, together with a ration of swede turnips and oat straw. Only one mild case of the disease (stiff-shoulder type) occurred in 1953. This experience, together with the observations of farmers and veterinary surgeons in the area, indicates that the feeding of grass silage to pregnant cows may be a useful way of reducing the incidence of the disease. Definite proof, however, has not yet been obtained.

A number of other possibilities of increasing the vitamin E intake suggest themselves. Thus, samples of wet distillers grains have been shown by Dr. Blaxter to contain 3·5 mg. total tocopherol per 100 grammes DM. As this feed is cheap and readily available in the area it may be that feeding about 20 lb. wet grains per day during the winter (equivalent to 140 mg. tocopherol) would be a useful supplement. Sufficient observations, however, have not yet been made.

Again, marrow stem kale, particularly the leaves, has a high tocopherol content and thousand-headed kale has a slightly lower content (Brown, 1953[3]). By growing a succession of kales and feeding them in winter and spring it is possible to have green food available through the period when grass is not available. Kale feeding may prove to be a valuable method of providing vitamin E in winter.

Arrangements for cows to calve from May onwards on grass would appear to be successful in preventing the disease, but the calves are not as valuable as early ones. Early calving (before New Year) is the ideal to be aimed at in breeding areas in the north of Scotland. It has the double advantage of producing calves of good market value, and the incidence of muscular dystrophy in the calves is very low.

On a few farms cows and calves go out by day and are housed at night throughout the winter. Many Highland and cross Highland cows are outwintered. So far only two cases have been observed in calves kept under either of these systems. It is worth recording that one of these was a calf born outside in March to an outwintered Hereford cow. On the farm concerned, in late April, the Aberdeen Angus cows and their calves which had been reared indoors in the traditional manner, were turned out into the same field as the outwintered cattle. Two days later four of the calves born inside and one of the calves born outside became typical shoulder cases of muscular dystrophy. It is presumed that all the calves indulged in unwonted exercise which precipitated the disease.

It is usual on farms where pedigree bulls are bred to feed some cod liver oil. A few cases of the disease have been encountered on such farms where feeding of the dams appeared to be sufficiently varied to render calf susceptibility to muscular dystrophy unlikely. These cases are considered to be induced by the anti-vitamin E effect of the unsaturated fatty acid fraction of cod liver oil (Blaxter, Wood & MacDonald, 1953[10]).

Summary

It has been established that a disease of suckled beef calves, which occurs in the north of Scotland when housed cattle are first put to grass in early summer, is muscular dystrophy. The resemblance of this disease to the description of "hairball" in the 1947 N.V.M.A. handbook of calf diseases and to descriptions of experimental muscular dystrophy in calves, is pointed out.

Many sudden deaths occurring in suckled beef calves in spring and early summer have been shown to be due to dystrophic lesions of heart muscle.

The disease can be prevented by ensuring that in-calf cows receive adequate quantities of vitamin E by mouth. It may also be prevented by dosing calves from birth with vitamin E, and suggestions are made on how feeding may prevent the disease. Treatment of early cases with vitamin E is effective.

Some field cases of muscular dystrophy induced by feeding cod liver oil have been enountered.

Acknowledgments.—I wish to extend my thanks to all practising veterinary surgeons and farmers in the north of Scotland who have freely made cases available for investigation and experiment. I also wish to thank Dr. Blaxter for his guidance and help and for permission to publish the photographs, which are all his. To Mr. P. L. Shanks I offer thanks for his encouragement and guidance.

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ATTESTATION IN STIRLINGSHIRE

Mr. W. Grant, Divisional Veterinary Officer, in his quarterly report to the Stirlingshire Public Health Committee, stated that every herd in the County had now had a second herd test and no premises remained outside the attested herd scheme. Stirlingshire county was ready for addition to the existing attested herd area, and he estimated that this would be accomplished in the case of the counties of Peebles, Dunbartonshire, Lanarkshire and Stirlingshire in the course of this year, probably about July. During the quarter under review, there had been only one reactor out of a total of 5,021 cattle tested.

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THE ASSOCIATION OF VETERINARY STUDENTS

The following Officers have been elected to the Council of the Association of Veterinary Students for the year 1954-55: President, J. B. Derbyshire (London); Vice-President, D. B. R. Poole (Dublin); Secretary, J. A. Brownell (Dublin); Treasurer, W. J. Handcock (Dublin); Editor of Journal, G. Hoban (Dublin).

"CHRONIC" COPPER POISONING OF SHEEP

BY

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The literature on copper poisoning is not very extensive either in man or in animals, and it contains a number of divergent points of view as to toxic levels of the element, clinical behaviour of affected subjects, and so on.

Most veterinary authors, however, appear to agree that both acute and chronic forms of copper poisoning do occur in animals and that both types can have a fatal outcome. Indeed, acute copper poisoning has been the subject of comment in veterinary literature for many years, and under study since at least 1912 when Theiler (1912) in South Africa conducted experiments upon it. Various authors including Rose (1933) in Australia, the writer in Britain (1942) and Udall (1947) in America have reported acute cases of poisoning in different species of animals under a variety of conditions.

So called chronic or delayed copper poisoning has, however, received less attention and was not fully documented until 1934 when Boughton & Hardy (1934) showed that some of the cases in sheep in the U.S.A. previously described as icterohaematuria and icterohaemoglobinuria were, in fact, poisoning due to long-continued ingestion of copper sulphate. Before that Schaper & Lutje (1931), in Germany, reported losses in sheep pastured in orchards which had been sprayed with copper sulphate, and Beijers (1932) published a somewhat similar report. Lafenetre, Monteil & Galtier (1935) described deaths in sheep in a vineyard in France which had been treated with a copper spray, and Weiman (1939) encountered cases of chronic copper poisoning in cattle maintained near a copper smelting works. Some 25 per cent. of these cattle were affected after grazing there for about six weeks. More recently, considerable attention has been given to the subject by Australian workers who contend that copper poisoning in sheep from eating weeds which normally contain high levels of the metal is quite common in their part of the world. Grahame, Albiston & Bull (1949) indicate, moreover, that clover pastures with a moderately high copper content may also be toxic under certain circumstances. Eden (1940), Dick (1952), & Muth (1952) have made special studies of the general subject.

The writer has recently encountered a case in which numerous sheep which were grazed on orchard pastures succumbed to copper poisoning, and feels that it may be useful to contrast the findings in this case with those generally accepted in the literature.

History and Symptoms

In most cases of chronic copper poisoning of sheep the history is of a number of deaths in a flock occurring in fairly rapid succession but under apparently rather mysterious circumstances. The flock health is good, but individual sheep suddenly become ill, stop grazing, and die within two or three days. Newsom (1952) records symptoms in chronic copper poisoning of sheep, of dullness, loss of appetite, weakness, staggering gait and convulsions. In affected animals he found that the pulse is rapid and weak, and the respirations hurried and shallow. The urine is red or coffee coloured. There is no temperature reaction. Newsom regards yellowish discoloration of the mucous membranes and of the skin as the most significant sign of the disease. The British Veterinary Codex (1953) lists loss of appetite, emaciation, weakness and staggering gait

followed by jaundice and haematuria as characteristic symptoms. Greig & Boddie (1942) mention muscular weakness and nervous symptoms as typical, and Nicholson (1945) cites oedema of the ears and loss of wool as appearing in a proportion of affected subjects. Boughton & Hardy (1934) found that affected animals die within a day or two of showing symptoms, these generally being anorexia, weakness, icterus and haematuria. In man, Sydney Smith (1931), cites malaise, debility, and gastro-enteritis with peripheral neuritis and degeneration, and atrophy of muscles in the later stages as typical of chronic copper poisoning.

In our cases, the circumstances were as follow. The flock, originally numbering about 120, ran on 85 acres of orchards, grazing each of 12 areas in turn. About a year before our examination was made 22 ewes and lambs had died from unknown causes. Some three months later a further 20 lambs succumbed from what was thought to be pulpy kidney disease. Specific inoculation had good results and losses ceased. Seven months after this, however, in mid-November, losses recommenced and, over a period of two and a half months, 21 of the remaining sheep died. In the main they displayed the symptoms described above except that there was never any visible sign of loss of weight, oedema of the ears, or loss of wool, and a number of animals which died showed no symptoms of jaundice or haematuria.

Enquiries revealed that the orchard had been sprayed with cuprous oxide in October and November, the material used being equivalent to 50 per cent. weight per weight of copper. This was employed at a dilution of 5 lb. per 100 gallons of water and applied at a rate of 400 gallons per acre, equivalent to 10 lb. of copper per acre. The 12 areas into which the orchard was divided were grazed in turn, the sheep occupying each area for one week. The spraying programme was so adjusted that sheep were not introduced into an area until a minimum of 14 days had elapsed since the last spraying. The first death occurred in November two weeks after all spraying had ceased, and two more sheep succumbed during that month. Eight died in December and 16 in January, some of them more than two and a half months after spraying had been completed.

Post-mortem Findings

Muth (1952) lists generalised icterus, yellow friable liver, enlarged extremely dark kidneys with a metallic lustre, and coffee-coloured urine as typical autopsy findings in chronic copper poisoning of sheep. Baum & Seeliger (1898) noted that there were often considerable differences in these findings between individual sheep. In only one case examined by them did they find general icterus. In all the others there was chronic catarrh of the small intestine, and swelling, inflammation, fatty degeneration, atrophy and necrosis of liver and kidneys. Nicholson (1945) mentions gastro-enteritis and liver degeneration as typical of these cases, and states that there may also be signs of generalised icterus and oedema together with myocarditis, nephritis and cystitis. Newsom (1952) and Boughton & Hardy (1934) found generalised icterus a constant feature together with intense darkening of the kidneys, swelling and degeneration of the liver, and enlargement and darkening of the spleen.

We were presented with three sheep for autopsy, one which had succumbed 24 to 36 hours previously, one which had marked signs of the disease when examined by us and which died three to four hours before the *post-mortem* examination was made, and one which showed no symptoms of disease over a period of observation and was killed immediately before autopsy was performed. The *post-mortem* examinations were made by Mr. W. T. Harrow, M.R.C.V.S. The sheep which had been dead for 24 to 36 hours proved to be decomposing rapidly and was unfit for detailed examination. Its liver and kidneys were completely degenerated. The carcase muscles and organs showed deep red discoloration but no signs of jaundice. Nor was there any yellowing of the skin or mucous membranes.

In contrast the carcase of the sheep which died only two to three hours before autopsy was deeply jaundiced. Its skin and mucous membranes were bright yellow; the subcutaneous fat was deep orange; and the muscles were dark red-brown. The general appearance of its flesh was more like that normally presented by the horse than by the sheep. The nutritional condition of the musculature and the body fat was excellent. There were no signs of weight loss or emaciation as noted by some authors, nor, from the evidence of rumen contents, had there been any loss of appetite until, at the most, a few days before death. Detailed examination of organs revealed a few pneumonic patches in the lungs, enlargement and deep engorgement of the kidneys, and marked degeneration of the liver which was almost completely broken down to a bright yellowish-brown pulp. The uterus contained two normal foetuses. The small quantity of urine which remained in the bladder was dark coffee-coloured.

The third sheep showed no gross abnormalities on autopsy. It was in excellent condition with ample subcutaneous and omental fat and had been eating up to the time it was killed. Liver, spleen, kidneys and bladder were normal on macroscopic examination.

In none of these animals were there any signs of gastro-intestinal irritation or of generalised oedema, nor were there any signs of myocarditis.

An interesting observation was made by the owner of the sheep at a later stage of the investigation. He attempted to salvage a number of animals, some of which were showing early signs of the disease, but including others which, according to him, were quite normal. At slaughter the carcases were of good quality and were acceptable to the slaughterhouse authorities; but they darkened in colour very appreciably after 24 hours and were all condemned as unfit for human consumption.

Biochemical Investigation

Chemical analyses were made of samples of liver, kidney, rumen contents and small intestine from sheep number 2, *i.e.*, the one which died a few hours before autopsy and of liver, kidney, rumen contents, small intestine and large intestine from sheep number 3, *i.e.*, the one which was killed and examined immediately afterwards. The results are set out in Table I. These figures are interesting in that the copper levels far exceed those which are accepted as normal in both the sheep which succumbed to the disease and that which was apparently quite normal when it was killed. Indeed, so far as the liver is concerned, the unaffected sheep had more copper in it than the one which died—655 p.p.m. against 630 p.p.m. on a wet weight basis. Significantly, however, the kidneys of the sheep which succumbed contained considerably more copper than those of the unaffected animal—three to four times as much, indicating perhaps that in the first sheep breakdown of the liver storage mechanism had occurred whereas in the second animal, despite the very high liver levels some control of excretion was still being exercised. There is also evidence from the figures that on release of large quantities of copper the main portal of excretion is through the kidneys. There is no unanimity of opinion as to the

Table I

Material		Liver		Kidney		Rumen		Small intestine		Large intestine	
		Sheep 2	Sheep 3	Sheep 2	Sheep 3	Sheep 2	Sheep 3	Sheep 2	Sheep 3	Sheep 2	Sheep 3
Total solids (dried at 100° C. to constant weight)		28·9 per cent.	29·9 per cent.	23·1 per cent.	15·6 per cent.	—	13·3 per cent.	—	11 per cent.	—	18 per cent.
Arsenic as p.p.m. AS_2O_3	*Dry weight* ...	1	Trace	—	1	—	1	—	1	—	1
	Wet „ ...	<1	1	<1	1	0·25	1	0·15	1	—	1
Copper as p.p.m. Cu	*Dry weight* ...	2,200	2,200	450	155	—	60	—	75	—	66
	Wet „ ...	630	655	110	24	13	7	5	8	—	12
Cobalt as p.p.m. Co	*Dry weight* ...	<1	<1	<1	<1	<1	<1	<1	<1	—	<1
	Wet „ ...										
Iron as p.p.m. Fe	*Dry weight* ...	520	217	850	280	—	2,000	—	3,000	—	3,600
	Wet „ ...	150	65	197	40	180	260	205	320	—	650
Lead as p.p.m. Pb	*Dry weight* ...	6	7	2·5	8	—	24	—	34	—	16
	Wet „ ...	1·8	2	0·6	1	<1	3	0·5	4	—	3
Zinc as p.p.m. Zn	*Dry weight* ...	62	128	69	100	—	70	—	94	—	98
	Wet „ ...	18	38	16	17	16	9	6·3	10	—	18

levels of copper in the liver of normal sheep. Eden (1940) gives 12 p.p.m. as a wet weight normal and Boughton & Hardy (1934) 16 p.p.m. Nicholson (1945) gives 25 p.p.m. as normal for the dog. All are agreed, however, that considerably greater amounts of copper can be contained by the liver without apparent ill-effect. Dick (1952) has recorded normals of up to 213 p.p.m. and Eden up to 290 p.p.m., but Muth (1952) regards levels of over 500 p.p.m. for normal wet weights as very rare indeed. It must be taken, therefore, that in both our cases where the wet weight levels exceeded 600 p.p.m., poisoning with copper was occurring. So far as other cases are concerned Eden (1940) records cases of poisoning at levels of 576 p.p.m. and Muth (1925) at 1,078 p.p.m. and 1,340 p.p.m.

For kidney levels Eden (1940) regards 5 p.p.m. as normal on a wet weight basis against our figures of 24 p.p.m. for the sheep which was killed and 110 p.p.m. for the sheep which succumbed to the disease. There is, however, a general paucity of analytical data in the literature, and in some cases no clear indication as to whether they are on a wet or dry weight basis, a point of considerable importance.

At the same time as the autopsies were made a fourth sheep was sent to us from the flock and grazed on a paddock whose herbage is known to be normal in copper content. Two days after arrival its faeces were examined with the results shown in Table II.

Table II
Parts per Million (Wet Weight)

Material	Arsenic	Copper	Cobalt	Iron	Manganese	Lead	Zinc
Faeces	<1	100	72	3,000	<1	4·3	264

This table shows high levels for copper, iron and zinc. The copper figure was somewhat surprising to us especially considering the relatively low bowel copper figures of the two other sheep. Moreover a cobalt level of 72 p.p.m. appeared to be very high when only traces of cobalt were found in the others. Nicholson (1945) makes the point that a high copper content in the faeces is very significant, but in this case the sheep concerned showed no signs of illness, continued to thrive, and is still alive.

Pasture Examination

An examination was then made of grazing samples from representative paddocks in the orchard in which the sheep had been run. Each sample comprised approximately 10 grammes of grass cut to within 1 inch of the soil level. One set of samples was taken two months after the completion of spraying and the second set five months after this time. The results are set out in Table III.

Table III

Paddock	Cu p.p.m. two months	Cu p.p.m. five months
1	150	10·5
2	80	—
3	160	15
4	250	10·3
Controls (average)	8	8

It is obvious from these figures that the sheep must have been ingesting amounts of copper which were considerably greater than normal. No doubt, too, the copper content of the herbage was higher soon after spraying than after an interval of two months.

Exactly how much copper the sheep did ingest is, of course, problematical. Taking the New Zealand figure of $2\frac{1}{2}$ to $2\frac{3}{4}$ lb. of dry matter as the average daily food consumption of sheep, grass intake should average about 12 to 13 lb. Even two months after spraying therefore, the daily intake of copper in our sheep must have been nearly 1 gramme which, by all accepted standards, is sufficient quite rapidly to produce toxic effects, especially in animals which already have a high liver content of the element. It is surprising in the circumstances that most, if not all, of the sheep did not succumb.

On this point Muth (1952) found that copper levels of 42 p.p.m. in herbage were highly dangerous to sheep. Clover samples taken in a peach orchard three months after spraying showed this level. Grahame and co-workers (1949) have shown that a clover pasture with a copper content as low as 12·8 p.p.m. can be toxic to sheep if they are maintained for a sufficient length of time upon it. Grahame has encountered cases where the addition of as little as 25 mg. of copper daily to the normal diet has

raised the liver copper to dangerous heights when practised over a period of 30 weeks. Eden (1940) found that 1 to 1·5 grammes of copper sulphate caused chronic poisoning in sheep when given for 30 to 80 days and Boughton & Hardy (1934) found 0·645 gramme of the same salt toxic when given daily for 41 days. In their cases two sprayings a year with Bordeaux mixture were given to trees which resulted in a yearly application of 44·4 lb. of copper sulphate per acre of pasture.

Physiological Effects of Copper

Copper occurs in all living matter of which it is an essential ingredient. Its exact function, however, is not fully determined. It would appear to have a catalytic action in many body reactions. Copper is, of course, essential for the utilisation of iron in haemoglobin formation but it is not part of the haemoglobin molecule nor of the framework of the erythrocytes in the circulation. It appears to play a rôle in enzyme actions. When given by mouth it is fairly quickly absorbed and is deposited in the liver and kidneys from which it is eliminated very slowly in the bile and urine. The liver is the main storage depôt for the element, and it is normally able to hold considerable quantities over several months. Usually it circulates in the blood at a level of about 0·2 mg. per cent. equally distributed between plasma and corpuscles. In "chronic" poisoning cases there is apparently a breakdown in the mechanism of storage due to severe damage to the liver and kidneys. Despite heavy intake of the metal by mouth over a long period the blood levels usually remain normal, but when liver breakdown occurs the level increases to 10 to 20 times normal and immediate symptoms of copper poisoning appear. The known pharmacology of copper therefore, supports our diagnosis of "chronic" copper poisoning.

Terminolcgy

It is perhaps appropriate to draw attention here to the point that the term "chronic" is something of a misnomer. From what has been said above it refers only to the time it takes to bring about symptoms. In sheep with small doses of copper, this may be as short as 20 days and as long as 100 days, depending on the copper status at the beginning of ingestion and possibly on other factors. When symptoms once develop, however, a very acute disorder is manifest and death results in one to four days, as in the cases we had under examination.

Discussion

It would appear from our cases and from a scrutiny of the literature that the general tolerance of sheep to copper is high, but that excessive doses of the element have a cumulative effect if given repeatedly. As is to be expected, individual animals vary considerably in their susceptibility to it. Some sheep succumb to levels which are apparently well tolerated by others. A feature of the terminal phase of the disorder is the rapidity with which it occurs. Sheep in apparently excellent thriving condition one day may be at or near death's door the next. This sort of picture does not usually feature in the clinician's mind as characteristic of chronic mineral poisoning. Nor is his task made easier by the fact that contamination of the pastures may only have occurred several months previously, apparently without direct relation to the cases on hand. In this respect there is no doubt that a careful analytical examination is of the greatest value in diagnosis. Possibly more analyses would reveal a greater number of cases of this nature than has been recorded to date.

A final point which should perhaps be stressed is that the circumstances of our cases may have been unusual in that for a period of about two months after spraying there was practically no rain. This probably meant that the cuprous oxide which is in any case specially formulated to render it persistent, was not washed off to any appreciable extent. Moreover, growth of the grass was severely checked by lack of moisture, which would tend to keep it high in copper content and make it dangerous to sheep grazing upon it.

From the fact that many sheep survived, despite the considerable intake of copper, it might be concluded that grazing the grass well down before spraying takes place, or keeping the sheep off the land until a fair amount of growth of grass takes place should obviate the trouble. Awaiting heavy rain after spraying before the re-introduction of sheep would also seem to be a sound precaution. It is in fact one already practised by some knowledgeable farmers.

Summary

An outbreak of "chronic" copper poisoning in sheep is described. The affected animals were pastured in an orchard which had been sprayed with a copper preparation. The outbreak had a number of unusual features which are contrasted with those recorded by other authors.

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AGRICULTURAL FOOD PRODUCTS, LTD.

Members of the Profession who have had association with this company, and with Vitamins, Ltd., may be interested to hear that a special party was given recently in honour of the 90th birthday of Mr. J. H. Wrentmore, Senior Director of both concerns. In proposing Mr. Wrentmore's health, the Chairman and Managing Director referred to the fact that Mr. Wrentmore had been responsible for marketing the wheat germ in the commercial form of Bemax with which his name would always be associated. It was mentioned that during the first World War Mr. Wrentmore served in a bomb disposal squad and took part in the removal of an unexploded bomb which threatened the Tower of London.

CLINICAL COMMUNICATION

TREATMENT OF CANINE DIARRHOEA

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Diarrhoea in dogs is an ever-present problem to the small-animal clinician. Whilst some cases respond readily to routine treatment, others tax his skill and patience to the full. It is in an endeavour to find a more effective routine treatment than the numerous astringent and other anti-diarrhoeic mixtures that this investigation has been undertaken.

For the purpose of the trial I divided canine diarrhoea into two rough aetiological categories which I classified as primary and secondary.

Secondary diarrhoea embraced those cases which were associated with another disease, for example, canine distemper. Primary diarrhoea, on the other hand, consisted of those cases which were not obviously associated with any other condition and which appeared to be localised in the bowel. I am aware that this classification is little more than empirical, but I feel that some distinction must be made. Obviously the chief concern in those cases where diarrhoea is secondary, must be the treatment of the underlying disease and whilst endeavours to arrest the diarrhoea are indicated, a clinical trial of a therapeutic agent would, in these circumstances, be meaningless. I have therefore restricted the trial to cases of " primary diarrhoea " and to those in which no concurrent treatment was used with the product under test. I have made no attempt to determine the precise aetiology of the cases treated as I feel that this enormous task is for the laboratory rather than for the clinician. The small animal practitioner is more interested in curing his diarrhoea cases than in determining their bacteriology. Furthermore, it is usually inadvisable to delay treatment whilst awaiting the results of lengthy bacteriological examinations.

In the past, I have used, with varying degrees of success, several of the sulphonamides which produce high concentrations in the intestinal tract. Recently, however, my attention was drawn to phthalylsulphacetamide which has been shown to be virtually non-toxic with minimal blood levels and, unlike other sulphonamides in contemporary use, readily penetrates the tissues of the intestinal wall which often harbour the seat of enteric infections. Phthalylsulphacetamide was stated by Rogoff to be the drug of choice in intestinal infections, whereas Christian reported that it proved to be a valuable treatment for infectious calf scours due to its ability to be absorbed into the intestinal lining, thus reaching deep-seated infections.

On the basis of this published information, I decided to utilise phthalylsulphacetamide in these trials. For convenience of administration I used a suspension of phthalylsulphacetamide (10 per cent.) which also contained two intestinal detoxicants—kaolin (10 per cent.) and pectin (2·5 per cent.) plus sulphathiazole (4·22 per cent.) a highly active absorbable sulphonamide, to combat any associated systemic complications resulting from the enteric infection. The dosage employed was on the basis of 1 fluid dram of the suspension per 15 lb. bodyweight twice daily.

Results of the trial are shown in the accompanying table. In general, the variation in breed and age represents a fair cross section of dogs seen in my surgery, and certainly no conclusions could be drawn as to any breed-incidence from this number of cases. The predominance of Alsatians is due to the treatment of a bitch (Case 4) and its litter of six puppies (Cases 5A-F). Anthelminthic treatment was given prior to the trial in Cases 3 and 5A-F but did not check the diarrhoea. Cases 4 and 5A-F were given a bowel sedative which again had no effect on the diarrhoea. None of the other cases received any previous treatment. Cases 12 and 15 did not respond to treatment. The former showed signs of chronic nephritis and was destroyed at the request of the owner, whereas Case 15 responded to oral streptomycin.

In every case diarrhoea was severe, but none showed any significant elevation of temperature. Anorexia was present only in Cases 3, 7, 9, 10, and 11. Appetite generally returned at about the time the diarrhoea ceased.

Although diarrhoea generally ceased after two to five days, I have, in order to minimise the risk of relapse, continued treatment for eight days in every case. It may well be, however, that a shorter period of treatment is adequate. This investigation has been proceeding for four months and, during this time, no toxic effects have been seen in any of the cases treated and, at the time of writing, no relapses have been reported.

Case No.	*Breed*	*Age*	*Approx. weight*	*Results*
1.	Cross-bred Terrier	1½ years	30 lb.	Diarrhoea persisted for three days then a gradual return to normal faeces.
2.	Boxer	6 months	50 lb.	Diarrhoea ceased in two days, no recurrence.
3.	Cocker Spaniel	10 weeks	3 lb.	Diarrhoea ceased on fourth day.
4.	Alsatian	2 years	60 lb.	Diarrhoea ceased on third day.
5A-F	Alsatian	6 weeks	10 lb.	Diarrhoea ceased between second to fifth day.
6.	Alsatian	6 months	60 lb.	No faeces for three days, then normal.
7.	Sealyham	18 ,,	20 lb.	Diarrhoea ceased on fifth day.
8.	Cross-bred Sheep dog	8 weeks	8 lb.	Diarrhoea ceased on third day.
9.	Cross-bred Terrier	8 ,,	6 lb.	Diarrhoea ceased on third day.
10.	Springer Spaniel	3 years	60 lb.	Diarrhoea ceased on third day.
11.	Cross-bred Terrier	4 ,,	15 lb.	Diarrhoea ceased on fourth day.
12.	Cross-bred Terrier	8 ,,	20 lb.	No response.
13.	Cairn	3 months	10 lb.	Diarrhoea ceased on sixth day.
14.	Labrador	6 ,,	50 lb.	Diarrhoea ceased on third day.
15.	Poodle	6 ,,	20 lb.	No response.
16.	Cross-bred Spaniel	2 years	40 lb.	Diarrhoea ceased on fourth day.
17.	Pekinese	5 ,,	15 lb.	Diarrhoea ceased on third day.
18.	Poodle	1 year	20 lb.	Diarrhoea intermittent for six days then ceased.
19.	Yorkshire Terrier	18 weeks	6 lb.	Diarrhoea ceased on third day.
20.	Cross-bred Terrier	12 months	20 lb.	Diarrhoea ceased on third day.

This work is continuing but I feel that the results already achieved are of sufficient interest to warrant publication.

SUMMARY

1. The problem of canine diarrhoea to the small animal clinician is described and the value and convenience of treatment to effect a cure without bacteriological examination is emphasized;

2. Twenty-five cases are described which were treated with a suspension containing phthalylsulphacetamide, sulphathiazole, pectin and kaolin;

3. Diarrhoea ceased after from two to five days of treatment in 23 cases; the remaining two showed no response. No toxic effects or relapses were observed;

Acknowledgments.—The material used in these trials (Sterathal Suspension) was supplied by Messrs. Ward, Blenkinsop & Co. Ltd.

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ABSTRACTS

The Provision and Maintenance of Laboratory Animals*

Sir Thomas Dalling once wrote, " Every scientist who correctly appreciates the part played by laboratory animals in research work recognises the importance of using the right technique in their maintenance and handling. Not only is the accuracy of our results at stake, but we are under an obligation to show the utmost consideration for the animals themselves. It behoves us, therefore, to see that they are produced and maintained under conditions that will conform to a high standard of humaneness and will at the same time ensure that our conclusions shall be thoroughly reliable." It was to further these aims that the Laboratory Animals Bureau, which sponsored and published this volume of " Collected Papers," was formed in 1947 by the Medical Research Council under the direction of Professor R. E. Glover.

There are five papers in this volume, and each is devoted to an important aspect in the provision and successful maintenance of laboratory animals. Dr. Lane Petter, who is the present Director of L.A.B., contributes the first paper in which the supply of laboratory animals is discussed. There are two main producers of the most commonly used animals, *viz.*, commercial breeders and the large user research institutes, while dogs, cats, and the more exotic animals are acquired through dealers. One of the services provided by the L.A.B. is that it acts as a link between users and suppliers. The growing importance attached to the provision of laboratory animals is shown by the fact that, since 1939, the number of laboratory animals used for research work has doubled. The second paper by A. N. Worden is a review of the dietetic requirements of laboratory animals. In particular the value of cubed diets, provided they are properly used, is emphasised.

The third paper focuses attention on the most important single factor in the welfare of laboratory animals, the animal attendant. A. Mundy, who is the Hon. Secretary of the Animal Technicals Association, discusses the status, training and duties of the people who have to carry much of the responsibility for the health of laboratory animals, upon which depends the success, or failure, of animal experiments.

One of the foundations of good animal husbandry is the prevention of disease, and the control of infection in small animal breeding units is the subject of the fourth paper, by J. Scarnell. The main points raised are that the breeding establishment should be completely separate from the small animal house, and strict precautions should be exercised to prevent the access of vermin, and vermin-contaminated foodstuffs. Each animal species should be confined to a separate room. Sickness and death among the animals should be immediately investigated, and the in-contact animals kept in quarantine until the cause is established, and the appropriate counter measures instituted.

The breeding of laboratory animals for experimental purposes is complicated by the fact that research workers make demands at irregular intervals for large numbers of animals of approximately the same age and weight. Dr. Paterson in the last paper offers valuable advice to those responsible for the maintenance of breeding establishments, and indicates how, by intelligent management procedures, it is possible to exercise some control over output, so that gluts and famines are partly avoided.

D. W. J.

* * * * * *

Survey of Leptospiral Infection Rates in Rats*

The authors carried out a survey of the leptospiral infection rate in rats in a valley in Wales. The farms concerned, mostly dairy farms, lay between the 250 and 750 ft. contour lines. The water supply was from springs and streams and there was no main sewerage, the streams receiving both domestic sewage and drainage from the farm buildings.

In all, 357 rats were examined. They were either trapped or poisoned and as they were not received fresh at the laboratory, the only feasible method of diagnosis was by histological examination of kidney sections stained by Levaditi. Leptospirae, assumed to be *L. icterohaemorrhagiae* were found in 23 per cent. of the rats. There was no difference in the sex incidence, but the infection rate was very low in young rats as compared with adults.

Serum from six workers on two farms in which the rat infestation was high, contained no *L. icterohaemorrhagiae* agglutinins, nor were there any known cases of human or animal infection in the whole area surveyed.

The authors suggested that some unknown factors were acting as a barrier to transmission from rat to man and to domestic animals without affecting the dissemination of leptospirae among the rats themselves. I.W.J.

* * * * *

Distemper Immunisation of Ferrets by Nebulisation with Egg Adapted Virus*

This paper describes distemper immunisation of ferrets by inhalation, using a 3-minute exposure to the *Onderstepoort* strain of egg-adapted distemper virus in the

*Laboratory Animals Bureau. (1953). *Collected Papers.* 1.

*Infection Rates of Rathis Norvegicus with *Leptospiraicterohaemorrhagiae* in Gt. Britain. I. A Rural Area in Carmarthenshire, Wales. BROOM, J. C., & GIBSON, E. A. (1953). *J. Hyg., Camb.* 51. No. 3.

*Distemper Immunisation of Ferrets by Nebulisation with Egg Adapted Virus. GORHAM, J. R., LEADER, R. W., & GUTIERREZ, JOYCE C. (1954). *Science.* 119. 125–126.

form of an aerosol. Ferrets were challenged with virulent virus at varying intervals of time after exposure to the aerosol. Details are given of the preparation of the virus suspension aerosol and the challenge virus.

Immunity was induced by the inhalation method, the onset of resistance occurring 4 to 5 days after exposure to the aerosol; it had been shown in a previous investigation that the lag period was two days when intramuscular inoculation was used.

A footnote states that, elsewhere, the aerosol immunisation technique has been used successfully in mink.

C. A. E. B.

REVIEW

[**Veterinary Pharmacology and Therapeutics.** By L. Meyer-Jones. The Iowa State College Press, Press Buildings, Ames, Iowa. Price $11.00.]

This is the best book on veterinary pharmacology and therapeutics so far produced. It is a large book, of 820 pages divided into 14 parts and 60 chapters.

Part I consists of eight introductory chapters concerned with basic details such as drug sources, prescription writing, weights and measures, drug forms and administration, such phenomena as synergism and cumulation, and, lastly, with the ways in which drugs may affect tissues.

The remaining parts deal with drugs grouped mainly according to the body system upon which they exert their actions, although there are a few which are not so linked. These, the antibiotics, sulphonamides, and anthelmintics, with possibly the " External Parasiticides and Antiparasitics," might have been adequately grouped together as Chemotherapeutics. A final part deals with lethal agents —rat poisons and methods of euthanasia.

This arrangement is very comprehensive and quite logical, being based, as the author points out in an interesting preface, on the " Program " evolved over many years for the teaching of veterinary students in Iowa State College, where he is professor of pharmacology.

Professor Meyer-Jones has chosen his material judiciously and has achieved his aim of " a progressive, middle-of-the-road approach," selecting wisely from the commonly accepted, the newly used, and also those drugs which are as yet hardly more than names to the British clinician. His method of presenting the features of these drugs is clear, and sub-headings are numerous; the English is excellent, with very few " Americanisms."

Part I is well presented, though a few more illustrative examples might be included with advantage. The second and third parts, comprising about 60 pages, and dealing with agents affecting skin and mucous membranes, and the alimentary system respectively, are rather scrappy with neither so many examples nor so much detail as has been accorded these " Systems " in the past. However, in Parts IV and V, which are allotted to central and peripheral nerve depressants (including anaesthetics), and stimulants, the author gets into his stride, the examples are numerous and detail is good.

The chapters on those most difficult drugs which affect the sympathetic and parasympathetic systems and are grouped in Part VI, as well as those similarly difficult drugs comprising Part VII which act on the heart, are dealt with efficiently, if a little academically.

The best chapters in the book are, as might be expected, those on the sulphonamides and antibiotics. These contain, in Parts VIII and IX, a comprehensive review of the actions and uses of all the common sulphonamides, the widely used antibiotics penicillin, streptomycin and chloromycetin, and also considerable information on those antibiotics not yet in common use for veterinary purposes in this country, terramycin and aureomycin.

Parts X and XI dealing with antiparasitic agents in the broadest sense are not quite up to the high standard of the preceding two parts, yet are still good, though it is disappointing to see the perpetuation of those old relics of nomenclature " Vermicides and Vermifuges." It is, however, interesting to see how the author, after defining these terms, gives a good example of the confusion which they encourage. Nematocides, he says, are agents which " destroy *or expel* " nematodes. Taenicides, similarly, are drugs which " destroy *or expel* " tapeworms!

Part XII is interesting in that it attempts a new approach to many " old drugs " by collecting them together under the heading of " Drugs influencing Tissue Metabolism." In these nine chapters are mentioned such diverse drugs as the electrolytes sodium and potassium, the sugars, mercurials, fat and water soluble vitamins, calcium and magnesium, the trace elements cobalt and manganese, the " Toxic Minerals " arsenic, lead, fluorine, and finally the thyroid, insulin, pituitary and adrenal cortex hormones. As would be expected, a completely successful integration and correlation of these drug actions is impossible with present-day physiological knowledge, but there is a considerable amount of valuable information presented. The same might be said of the part dealing with the reproductive system drugs.

The adverse impressions which remain after reading this book are not numerous. Perhaps something smaller would be more portable, and to this end the author might have been more ruthless in his editing, thereby cutting out irritating and needless repetition—some sentences are repeated almost word for word after only short intervals. Comprehensive and excellent references are drawn from world-wide publications, including many British ones, but are so many reference interpolations in the text necessary? They distract the reader and tend, by their number, to create an impression of lack of personal conviction and experience, rather than the correct one of extensive acquaintance with modern discovery. Perhaps more positive therapeutic indications might be incorporated in chapters other than those concerned with chemotherapeutics which are very good in this respect. Factual errors are rare though some readers may, inevitably, find assertions with which they cannot agree, but which, nevertheless, do not materially affect the high standard of the work.

The binding is excellent and presents an initial impression of quality which is fully supported by the text.

The universal appeal of this fine book should ensure that British students and clinicians, teachers and research workers, will all be grateful to Professor Meyer-Jones and his publishers for providing this milestone in veterinary literature.

REPORT

Colony of Trinidad and Tobago—Report of the Director of Agriculture for the Year 1952

This report, which embodies a section on animal health, is a wide survey of the agricultural work of the department.

The livestock population of the colony is not given, but in any case it is inadequate to meet the local needs for meat, and large quantities of frozen meat and mutton are imported from Australia and New Zealand.

The diseases listed as occurring in the colony are fowl cholera, Johne's disease, Newcastle disease, paralytic rabies, swine fever and tuberculosis.

By far the most important animal disease in the colony is paralytic rabies—a bat-transmitted disease. During the year 99 cases were reported, but this number does not represent the true incidence. A vigorous campaign of protective vaccination is in progress against this disease and despite the apathy of stockowners some 4,364 cattle, 85 water buffaloes, 2,208 horses, 508 goats and 39 sheep have been inoculated.

In reviewing the history of the disease the Director states that it first appeared in the colony in 1925 but was not diagnosed until 1931. Since the first appearance of paralytic rabies its incidence has averaged 1,000 cases a year, mainly in the rainy season. Transmission is by the vampire bat. This animal is unique in that it is the only known mammal which can not only survive infection with rabies virus, but also remain a source of virus probably for its entire life.

Measures against the disease are not confined to the destruction of bats but rely mainly on an active scheme of protective vaccination. The vaccine at present in use is a phenolized emulsion of brain tissue, but a modified avianised (live virus) vaccine is being tested.

A disease of poultry hitherto suspected to be either fowl typhoid or fowl cholera has now being diagnosed as Newcastle disease. It seems to be fairly widespread in the colony and an intra-nasal vaccine is being used.

Tuberculosis is another disease of substantial importance and occurs in all classes of livestock. The highest incidence occurs in water buffaloes, and reactors to the tuberculin test have ranged from 19 to 28 per cent. In cattle the percentage of reactors to the test is 1·4 to 2·4. Dairy goats also show a high incidence and a figure of 12·2 per cent. reactors to the tuberculin test is recorded. A peculiar feature of the disease in buffaloes is their ability to maintain condition and appetite even while in an advanced stage of infection. This feature, however, is not peculiar to water buffaloes. It is a prominent and constant characteristic of the disease in certain breeds of African cattle.

An outbreak of foot and mouth disease (A5 type virus) occurred in Martinique, French West Indies, and strong measures were taken to prevent its entry into Trinidad.

A disease thought to be necrotic enteritis occurred chiefly among young pigs. Internal parasites continue to give serious trouble among sheep, goats and young calves and deaths are numerous.

The head of the Animal Health division of the department, L. R. Hutson, B.V.Sc., D.V.Sc., has five veterinary officers to assist him.

W. W. H.

QUESTIONS IN PARLIAMENT

Foot-and-Mouth Disease

Mr. Deedes (May 6th) asked the Minister of Agriculture when he expects to receive the report of Sir Ernest Gower's committee on foot-and-mouth disease.

Sir T. Dugdale: I understand that the committee have nearly completed their report and hope to submit it to me by the end of June.

Mr. Hurd asked the Minister of Agriculture if the cause of the outbreak of foot-and-mouth disease amongst pigs at Aldermaston last week has been established; and if the particular source of origin has been identified.

Sir T. Dugdale: The source of the virus infection in this outbreak of food-and-mouth disease has not been definitely established. It is suspected to lie in uncooked swill stored on the infected premises, and that contamination was conveyed indirectly, probably by rats.

Warble Fly (Irish Cattle)

Mr. Hurd (May 6th) asked the Minister of Agriculture if he is aware of the arrival of large numbers of Irish cattle showing heavy infestation with the grubs of the warble fly; if he has yet received a report from his inspector at Fishguard on a badly diseased consignment which was shipped from Waterford last week; and what action, in conjunction with the Irish Department of Agriculture, he is taking to prevent recurrences which conflict with his Department's campaign to eradicate this pest.

Sir T. Dugdale: I am aware that Irish cattle entering this country are frequently found to be infested with warble fly maggots. Reports from my veterinary officer at Fishguard indicate that the proportion of heavily infested cattle in the consignment from Waterford mentioned by my hon. Friend was above the average for the season. I have had under review the whole problem of warble infestation to see whether it is practicable to take any further corrective steps, and I am writing fully to my hon. Friend.

NOTES AND NEWS

Diary of Events

May 15th.—Meeting of the Biochemical Society in the Department of Physiology, The University, Bristol, 8, 11 a.m.

May 19th.—Meeting of the Royal Society of Medicine, Section of Comparative Medicine, at the Society's House, 1, Wimpole Street, W.1, at 5 p.m.

May 19th.—Spring Meeting of the Eastern Counties Division, B.V.A., at the Bedford Lodge Hotel, Newmarket, 11.45 a.m.

May 20th.—Ordinary General Meeting of the South-Eastern Division, B.V.A., at the Royal Star Hotel, Maidstone, 2.30 p.m.

May 20th.—Meeting of the Sub-committee revising the B.V.A. Calf Report, at 7, Mansfield Street, W.1, 11 a.m.

May 26th.—Meeting of the Southern Counties Division, B.V.A., at the Polygon Hotel, Southampton, 6.30 p.m.

May 26th.—Annual General Meeting of the Society of Women Veterinary Surgeons. (See notice for details.)

May 27th.—General Meeting of the Herts and Beds Division, B.V.A., at the George Hotel, Luton, 7.30 p.m.

May 27th.—Meeting of the Sub-committee *re* Nutrition Conference at 7, Mansfield Street, W.1, 11 a.m.

May 27th.—Meeting of the Technical Development Committee at 7, Mansfield Street, W.1, 2.15 p.m.

May 28th.—Ordinary Meeting of the Royal Counties Division, B.V.A., at the Bull's Head Hotel, Aylesbury, 2.30 p.m.

June 1st.—One hundred and eleventh Annual General Meeting of the Royal College of Veterinary Surgeons at 10, Red Lion Square, London, W.C.1, at 12 noon.

June 1st.—Annual General Meeting of Victoria Veterinary Benevolent Fund at Royal College of Veterinary Surgeons, 10, Red Lion Square, W.C.1, to follow the above meeting.

June 9th.—Meeting of the Scottish Metropolitan Division, B.V.A., at the Minden Hotel, Peebles, 2.30 p.m.

June 10th.—President's invitation Luncheon and General Meeting of the Midland Counties Division, B.V.A., at the Penns Hall Hotel, Sutton Coldfield. Luncheon 1 p.m., Meeting 2.30 p.m.

June 11th.—Annual Dinner of the R.A.Y.C. Officers Club at the Grosvenor House, Park Lane, London, W.1.

Sept. 19th—25th.—72nd Annual General Meeting and Congress of the British Veterinary Association at Torquay.

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R.C.V.S. Examinations

Thursday, June 10th.—D.V.S.M. Written Examinations commence at Edinburgh.

Monday, June 14th.—D.V.S.M. Orals and Practicals commence.

Thursday, June 24th.—M.R.C.V.S. Written Examinations.

Friday, June 25th.—Ditto.

Monday, June 28th.—M.R.C.V.S. Practical and Orals commence (Panels " A " and " B ").

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PERSONAL

Births.—Bennett.—On April 13th, 1954, at the Cottage Hospital, Aberfeldy, to Euronwy, wife of D. G. Bennett, B.Sc., M.R.C.V.S., a son—Richard Lloyd.

Campbell.—On May 3rd, 1954, at Victoria Hospital, P.M.U., Keighley, to Elizabeth (*née* Caldwell), wife of Donald Campbell, M.R.C.V.S., of " Lydstep," Station Road, Oakworth, Nr. Keighley, a son.

Henderson.—On April 29th, 1954, to Mary (*née* Hawkins), wife of John F. Henderson, M.R.C.V.S., of Dorchester, a brother for Richard—Nicholas Fraser.

Forthcoming Marriage.—Hunter—Alexander.—The engagement is announced between Francis Edgar Hunter, M.R.C.V.S., younger son of the late Mr. R. Hunter and of Mrs. G. V. Hunter, 10, Chesterfield House, South Audley Street, London, W.1, and Carolyn Beverley, younger daughter of Mr. and Mrs. A. J. Alexander, 3, Dorchester Avenue, Cardiff.

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HONOUR FOR MR. W. J. B. ROBSON

His many friends will wish to congratulate Mr. Robson on his recent election as Provost of Laurencekirk. He is a former baillie having served eight years on the town council. On this occasion he topped the poll. His father, Mr. H. W. Robson, was a former Provost. Our readers will recall that Mr. W. J. B. Robson was Chairman of the Provisional Committee for last year's Aberdeen Congress.

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HOSPITALITY REQUIRED

Would any member be good enough to receive in his home during the month of July the 17-year-old son of a French veterinary surgeon living at Affreville, Algeria. The boy has been learning English for five years at school and wishes to practise the language in England in preparation for his school leaving examination next year. His father wishes to pay all expenses, as he feels he cannot offer return hospitality to an English boy or girl because of the climate of Algeria.

An Austrian woman student of the Veterinary College, University of Vienna, has written asking if she could see practice both in England and Scotland during the summer vacation this year, between July 15th and September 15th. In return for hospitality would be willing to help in any way she could.

Members who may be interested in either of these suggestions are asked to write to the General Secretary, B.V.A., 7, Mansfield Street, London, W.1.

MEETINGS OF THE ROYAL SANITARY INSTITUTE

During the month of June, the Royal Sanitary Institute announces the following meetings to be held in London. On June 4th, at 2.30 p.m., Alderman J. H. Mathews, M.A., J.P., of Southampton, and Mr. W. Brennan De Vine, M.C., F.R.C.V.S., D.V.S.M., a Vice-President of the Institute, will present papers on *The Interim Report of the Interdepartmental Committee on Slaughterhouses (England and Wales).*

Dr. H. C. Maurice Williams, O.B.E., Chairman of the Council of the Institute, will preside.

On June 9th, at 2.30 p.m., Mr. Stewart Forrest, M.R.C.V.S., D.V.S.M., Senior Veterinary Officer, Birmingham, will give a paper on *A Suggested National Meat Inspection Service.* Sir Weldon Dalrymple-Champneys, Bt., D.M., F.R.C.P., a Vice-President of the Institute, will preside.

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ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax:

Argyll.—Achrackman, Talbert (May 6).

Ches.—Arclid Cottage Farm, Arclid, Sandbach (May 3).

Cumberland.—Bolton Park, Mealsgate, Carlisle; Scarrowmanick Farm, Croglin, Carlisle (May 4).

Dumfries.—Almagill Farm, Dalton, Lockerbie (May 5).

Kirkcudbright.—Millhall Field, Upper Senwick Farm, Borgue (May 3).

Lanark.—Scorrieholme, Lesmahagow (May 5).

Lancs.—Duckworth's Farm, Ashworth, Bamford, Rochdale (May 3).

Notts.—20, Gateford Avenue, Worksop (May 3).

Yorks.—Coppice Farm, Chatsworth Grove, Harrogate (May 6).

Fowl Pest:

Beds.—New Farm, Colesden (May 5).

Lancs.—The Bungalows, Station Road, Little Hoole, Preston (May 2); Heatherlea, St. Michaels, Preston (May 3); Israels Farm, Rosemary Lane, Over Hulton, Bolton (May 4); Tarnacre Hall Farm, St. Michaels, Preston (May 5).

Norfolk.—Old Hall, Reymerston, Norwich (May 3).

Suffolk.—Tyrrells Farm, Stuston, Diss (May 5).

Surrey.—Ashton Hope, Felbridge, East Grinstead (May 4).

Sussex.—Cuttinglye Poultry Farm, Crawley Down, Crawley (May 5).

Yorks.—231, High Greave, Ecclesfield, Sheffield (May 6).

Swine Fever:

Cambs.—York Street Allotments, Cambridge (May 3); Sutton Road, Leverington, Wisbech (May 4); 45, Centre Road, Downfields, Soham, Ely (May 5); 13, Station Road, Manea, March (May 6).

Ches.—Thorn Tree Farm, Hyde Road, Bredbury, Woodley, Stockport (May 3); Thornton Green Cottages, Thornton le Moors, Chester (May 4); Holly Tree Farm, Lower Withington, Macclesfield; Common Farm, Whitegate, Northwich; Pipers Lane, Heswall, Wirral (May 5); Gandys Brook Farm, and Pastures Farm, Gawsworth, Macclesfield (May 6).

Derby.—Hollybank Farm, Scropton, Derby (May 3); Setcup Lane, Land Settlement, Eckington (May 5).

Herts.—Foxley Hotel, Bishops Stortford (May 3).

Lancs.—Bank Nook, Radnor Drive, Marshside, Southport (May 3).

Leics.—Main Street, Frolesworth (May 3).

Norfolk.—Jarvis Farm, Felbrigg, Roughton; Grove Farm, Tivetshall St. Mary, Norwich (May 3); Steel Drove, Hilgay (May 4).

Salop.—4, Broad Oak, Shrewsbury (May 3); The Lodge, Sundorne, Hadnall, Shrewsbury (May 5).

Suffolk.—Ringsfield Hall, Ringsfield, Beccles (May 3).

Sussex.—The Poplars, Tangmere, Chichester (May 3).

Wilts.—Green Lane Farm and New Venture Farm, Trowbridge (May 3).

Yorks.—29, Sproatley Road, Preston, Hull; Douglas Road Allotments, Parkwood Springs, Sheffield, 3 (May 4).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

CRYSTAL VIOLET VACCINE ON INFECTED PREMISES

Sir,—It is surprising that the letter from G. C. Lancaster on the use of crystal violet vaccine on infected premises (*Vet. Rec.*, April 24th, 1954) has passed without comment to date. We are therefore prompted to offer our observations on this point.

We have used crystal violet vaccine in this practice on infected premises where the owner is particularly desirous of maintaining a nucleus of his breeding stock. Such vaccination has been carried out in several confirmed outbreaks and also in unconfirmed cases, where insufficient animals were allowed to die and produce the ulcers and haemorrhagic kidneys required by the Ministry of Agriculture and Fisheries.

Where swine fever is present the owner has the following courses open to him, after having sent all fat pigs in for immediate slaughter: (*a*) Launch forth on an extensive course of serum treatment, (which we do not advise, although likely to prove of greater benefit to us than to the owner). (*b*) Sit tight and watch most of his pigs die off. (Some old sows take an awfully long time.) (*c*) Send all his pigs for slaughter; or (*d*) Vaccinate with crystal violet vaccine all remaining healthy stock.

The latter course we recommend, but before undertaking this line of action, it is essential that the owner accepts that serum treatment is of doubtful value and gives protection (if at all) for a very transient period and is also very costly. (If the serum should give some protection for a period of two to three weeks, we fail to see what advantage has been gained at the end of that period.) The owner is also informed that the use of the vaccine on infected premises is contrary to Ministry instructions, and that although we have been fortunate with this policy so far we may not continue to be so.

The objection to the use of the vaccine on infected premises is not easily understood unless it is that the Ministry fears that this course might bring discredit to their vaccine. Or is it that the use of the vaccine in these circumstances is likely to produce carriers, although our experience does not lead us to think so? If an animal which is incubating the disease is vaccinated, then death may be advanced a few days and nothing has been lost but the cost of the vaccine, since, clearly, if an animal becomes naturally infected before the vaccine has become effective it is likely that it will die whether it has been vaccinated or not.

Below are listed some cases of outbreaks where we have employed crystal violet vaccine. It is regretted that more cannot be given, but only confirmed cases in which figures are available have been noted here. We would point out that these are otherwise random cases and not selected because of the success of the policy pursued. In none of these cases was any swine fever prophylaxis previously carried out.

(1) *Autumn* 1951—*Herd A*

During a period of two months the pig population on this farm was reduced from 300 animals to 61 animals by death and casualty slaughter. Not more than 50 to 60 of the animals slaughtered would show a profitable return to the owner. In the early part of the outbreak only older sows were affected, but subsequently pigs of all ages became affected. The remaining 61 pigs comprised sows, two adult boars and young boars and gilts. These animals were all vaccinated with crystal violet vaccine and no further deaths ensued, although occasional deaths had occurred in all groups up to the time of vaccination.

(2) *Summer* 1952—*Herd B*

Two suckling pigs were ill for 12 days, and on *post-mortem* showed swine fever. All stores were sent for slaughter, and vaccination of all the remaining stock was carried out before confirmation was received by the Ministry: 16 sows, 19 gilts, and 80 weaners and sucklers were vaccinated. Several of the sucklers from the first affected litter, and an adjacent litter, were showing some symptoms at the time. No adult stock died or showed clinical symptoms after vaccination, but about 40 of the weaners and sucklers died including the whole of the litter in which the two original cases occurred. The whole of the litter vaccinated at the age of two weeks survived, although these were in an isolated pen. Some deaths from swine fever occurred in the young pigs as late as six weeks after vaccination.

All reasonable precautions were taken and no subsequent litters born on the premises while infection was present became infected. These litters were vaccinated at the age of one month and continued to remain free from swine fever.

(3) *Summer*, 1953—*Herd C*

Two gilts and one boar were treated with sulphamezathine for scouring of unknown aetiology and subsequently recovered. Six weeks later swine fever was confirmed in suckling pigs. Breeding stock of 11 was vaccinated and none developed swine fever. None of the sucklers was vaccinated and none survived. There was no further outbreak in subsequent litters.

(4) *Autumn*, 1953—*Herd D*

In another case known to us where serum treatment had been carried out the breeding stock was reduced from some 40 animals to 10, losses having occurred over a period of some weeks. No deaths were reported within one month of the remaining 10 having been vaccinated.

To date no further trouble has been experienced from swine fever in these herds. We conclude that vaccination of healthy breeding stock on infected premises is of considerable value. Vaccination of young pigs exposed to infection is less effective and tends to prolong the course of the disease (which suggests that the vaccine may have therapeutic as well as prophylactic properties).

Like Mr. Lancaster, we should like to know if and/or why this policy is wrong.—Yours faithfully, F. G. Lawfield, R. Hanscomb, Long Stanton, Cambs. *May 3rd*, 1954.

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THE GREYHOUND RACING ASSOCIATION

Sir,—With reference to the personal Note which appeared in last week's issue concerning my appointment as a veterinary surgeon to the Greyhound Racing Association, I hasten to make it quite clear that I am one of a group of our colleagues who act for that Company under the direction of Mr. James Bateman. —Yours faithfully, H. Kirk, "Croyland," 658, Finchley Road, Golders Green, N.W.11. *May 9th*, 1954.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period	Anthrax	Foot-and-mouth	Fowl Pest	Sheep Scab	Swine Fever
1st to 15th April, 1954	17	—	34	—	61
Corresponding period in—1953	32	2	9	—	217
1952	16	16	27	—	74
1951	22	—	12	—	38
1st January to 15th April, 1954 ...	88	11	445	—	396
Corresponding period in—1953	326	24	192	—	780
1952	135	72	92	1	349
1951	168	17	481	4	248

Tuberculosis Order of 1938

The number of bovine animals slaughtered under the Tuberculosis Order of 1938 during the period January 1st to March 31st, 1954, was 502.